tal is in operation, it is to be hoped that the highest standards of service will be emulated, to the end that hospitals everywhere shall become real health centers, both as regards curative and preventive medicine. With the aid of well organized hospitals, scientific medicine will move forward in continued betterment.

GOOD WISHES TO THE NEW EDITORIAL STAFF

The report of your present Editor appeared in the "Pre-Convention Bulletin" on page 210. Mention was made therein that your Editor will give up editorial supervision of CALIFORNIA AND WESTERN MEDICINE with this May issue. Also as there stated, the present Editor has had the great privilege of acting in that capacity since March 19, 1927, a period of twenty years lacking one. It has been a great pleasure to have been permitted to have had the editorial management of the OFFICIAL JOURNAL of the California Medical Association during these two decades.

Your retiring Editor, who will now take up his new duties as Honorary Historian, is under great obligation to the many members of the California Medical Association who have given to him their generous coöperation, and in making his farewell as Editor, he desires again to express his sincere appreciation of their kindly help.

It follows also that the best of wishes are tendered to those whom the Council will place in charge of CALIFORNIA AND WESTERN MEDICINE, and also as always, sincerest good wishes to all members of the California Medical Association.

EDITORIAL COMMENT†

NUTRITIONAL HYPERSUSCEPTIBILITY

A hitherto unsuspected nutritional factor causing hypersusceptibility to experimental pneumococcal infection in mice is described by Hitchings[1] and his associates of the Welcome Research Laboratories, Tuckahoe, N. Y.

The New York investigators maintained groups of white mice on three commercial diets: A, Purine Fox Chow; B, Rocklands mouse diet, and C, Robinson and Siegel's improved stock diet.[2] Before inoculation, certain groups were changed to a synthetic basal diet, consisting of casein, salts, cotton-seed oil, cod liver oil and sucrose, supplemented by thiamin chloride hydrochloride, riboflavin, pyridoxine chloride, calcium pantothenate, nicotinamide, p-aminobenzoic acid, and several other essential food factors.

Five days after this change, each mouse of all groups was injected intraperitoneally with 0.5 cc. of a 10^6 dilution of a 17 hour broth culture of pneumococci. The actual number of organisms injected varied from 235 to 700, as determined by plate count. Of the 56 mice on commercial diet A, 50 died within 6 days, a mortality rate of 89 per cent. The mortality was 60 per cent in the group fed diet B, and 70 per cent with diet C. Among 52 mice on the new synthetic diet, 45 survived, a mortality rate of but 14 per cent.

Even more spectacular results were recorded with mice injected with 10 times the routine test dose of pneumococci. In the group on diet A, all mice were dead by the third day, a mortality rate of 100 per cent. In a parallel test of mice fed for five days on the new synthetic diet, the mortality rate was but 22 per cent.

Hitchings believes that the most satisfactory explanation of the results is the assumption that some unknown factor stimulating the rate of multiplication of pneumococci is present in the commercial diets and absent from the synthetic basal diet. This hypothetical substance would not be essential for the mouse. The substance would be present in minimal concentration in the blood and body fluids except in so far as higher levels are maintained by a more or less continuous supply from exogenous sources. This would explain the rapid loss of nutritional hypersusceptibility when commercially fed mice are changed to the new synthetic diet. Attempts are being continued to identify and isolate this hypothetical bacterial growth stimulant.

A second possible explanation is suggested by the "opposition factor" or "anti-immunity protein" recently described by Day[3] of the Pathological Institute, St. Mary's Hospital, London. This haptin is semi-specific for the pneumococcus, and is found in numerous saprophytic bacteria, presumably in bacterial food contaminants. It is theoretically possible that this non-antigenic bacterial contaminant in commercial foods may cause a continuous depression of normal pneumococcus resistance, or a "blocking" antibody[4] of sufficient titer to cause the observed increased mortality in mice maintained on such commercial diets.

Either suggested possiblity would be of basic clinical interest.

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REFERENCES

1. Hitchings, G. H., and Falco, E. A., Proc. Soc. Exp. Biol. and Med., 61:54 (Jan.), 1946.
2. Robinson, H. J., and Siegel, H., J. Infect. Dis., 75:127, 1944.
3. Day, H. B., J. Hyg., 43:330, 1943.
4. Wiener, A. S., Proc. Soc. Exp. Biol. and Med., 56:173, 1944.

† This department of CALIFORNIA AND WESTERN MEDICINE presents editorial comments by contributing members on items of medical progress, science and practice, and on topics from recent medical books or journals. An invitation is extended to all members of the California Medical Association to submit brief editorial discussions suitable for publication in this department. No presentation should be over five hundred words in length.

A people [Americans] who are still, as it were, but in the gristle, and not yet hardened into the bone of manhood.

—Edmund Burke, *Conciliation with America.*

I have lived enough, for I die unconquered.

—Epaminonads. (Cornelius **Nepos**, *Lives: Epaminondas.*)